PostScript

LETTERS

Progressive retinal dysfunction in diffuse unilateral subacute neuroretinitis

Diffuse unilateral subacute neuroretinitis (DUSN) is a progressive retinal disorder related to subretinal migration of a nematode worm, usually occurring in healthy individuals.¹⁻³ It was reported first in North America and subsequently in South America and Asia.⁴⁻⁵ Different species of nematode have been identified,⁶ with a larger worm identified as a common parasite of raccoons (*Baylisascaris procyonis*), and a smaller worm causative of DUSN not yet characterised taxonomically. The early phase of the disease may show round yellowish outer retinal lesions with retinal pigment epithelium (RPE) changes and possible mild vitritis or papilloedema.¹⁻³ Late stages show multiple chorioretinal atrophic lesions, vessel narrowing, and disc pallor. Differential diagnoses include multifocal choroiditis and other "white dot syndromes." Careful and repeated examination may be necessary to detect the worm. Treatment relies on identification of the worm during funduscopy and direct thermal coagulation. Recently, a series of

patients have shown benefit when treated orally with the anthelmintic agent, albendazole.⁷

We report DUSN in a young boy in whom progressive electrophysiological deterioration occurred over a 4 year period before the worm was detected.

Case report

A healthy 9 year old boy, born in the United Kingdom to white parents, presented with painless right eye visual acuity (VA) loss discovered incidentally. There was no relevant medical or family history. He had been previously resident in Thailand and south east India and had spent summer holidays in the United States 2 years earlier. Best corrected VA was hand movements right; 6/5 left. A right afferent pupillary defect was noted. No sign of inflammation was seen. Right eye fundus showed RPE disturbance in the macular and peripapillary areas and moderate temporal disc pallor with no clear diagnostic features. The left fundus was normal (fig 1A). At the 1 year review his right eye had deteriorated subjectively but VA remained unaltered. Fundus appearance continued to show macular and peripapillary RPE disturbance. At the 4 year review, there were additional right eye yellowish lesions,

suggesting acute inflammation, with disc pallor and narrow vessels. A small nematode worm, of at most 500 µm, was detected on review of fundus colour photographs (fig 1B). Subsequent attempts to find the worm during examination were unsuccessful.

Electrophysiology was performed on three occasions incorporating the recommendations of the International Society of Clinical Electrophysiology of Vision (fig 2).⁸⁻¹⁰ The left eye was normal throughout. At presentation (fig 2A), right eye pattern visual evoked potential (VEP) was almost extinguished. The flash VEP was delayed. Right eye pattern electroretinogram (PERG) showed P50 reduction in keeping with marked right macular dysfunction but insufficient to explain the VA. ERGs were of low normal amplitude, with reduced b:a ratios relative to the left eye. These findings suggested involvement of both the optic nerve and retina. At the 1 year review (fig 2B), right eye PVEP and PERG were undetectable. Full field ERG showed inner retinal dysfunction with a reduced rod ERG, an electronegative maximal ERG with relatively preserved a-wave, and reduction in all photopic ERGs. There was reduction in ON-response b:a ratio with less OFF-response involvement. S-cone ERGs were subnormal. At 4 year review (fig 2C), right ERG showed progressive inner retinal dysfunction (b-wave) with additional photoreceptor involvement (a-wave).

Comment

This report documents progressive electrophysiological deterioration in DUSN. The ERGs suggest predominantly inner retinal dysfunction with later photoreceptor involvement. There was also optic nerve dysfunction, probably preceding retinal involvement and contributing to the poor VA at presentation. We have been unable to trace previous reports of VEP in DUSN or of sequential ERGs.

Inner retinal involvement was suggested by Gass³ who documented 36 cases with varying severity, late stage patients showing reduced ERG b:a ratio. None had ERG extinction, and the unaffected eye was always normal. One histological report, 15 months after onset, showed non-granulomatous vitritis, retinitis, and retinal and optic nerve perivascularitis,¹ but insufficient to explain the poor VA. A direct toxic effect of worm byproducts on inner and outer retina was proposed.¹⁻³ Another possibility is a secondary inflammatory or autoimmune process. Our observations are consistent with predominantly inner retinal dysfunction but are not mechanism specific. Further, possible effects on optic nerve function appear confirmed by the VEPs.

To conclude, our case exhibited progressive retinal dysfunction. The case demonstrates the utility of careful fundus photography in detecting these small worms and suggests an indirect toxic or inflammatory effect on inner retinal and optic nerve function, with relative preservation of photoreceptors.

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Figure 1 Colour fundus photographs: (A) at presentation, right eye (RE) fundus showing pigmentary disturbance in the macular and peripapillary areas and moderate temporal disc pallor. The left fundus (LE) is normal; (B) at 4 year review, showing retinal yellowish lesions, sign of previous inflammation, narrow vessels, pigmentary disturbance and disc pallor; a small nematode is visible in the right eye (C). The left fundus (LE) remains normal.

Figure 2 Pattern visual evoked potential, full field, and pattern electroretinograms. The findings from the left eye were normal throughout (see traces (C) for the left eye). (A) At presentation, right eye pattern visual evoked potential (VEP) was almost extinguished. Right eye pattern electroretinogram (PERG) showed P50 reduction in keeping with marked right macular dysfunction but insufficient to fully explain the level of VA. ERGs were of normal amplitude, with slightly reduced b:a ratios relative to the left eye. These findings suggested dysfunction of both the optic nerve and central retina. (B) At 1 year review, right eye PVEP and PERG were undetectable. Full field ERG showed increasing inner retinal dysfunction with a reduced rod ERG, an electronegative maximal ERG with relative sparing of the a-wave but some reduction, and reduction in all photopic ERGs. There was reduction in ON-response b:a ratio with less OFF-response involvement. S-cone ERGs were subnormal. (C) At 4 year review, right ERG showed progressive inner retinal dysfunction (b-wave) with additional photoreceptor involvement (a-wave).

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Neonatal methicillin resistant *Staphylococcus aureus* conjunctivitis

Methicillin resistant *Staphylococcus aureus* (MRSA) is an infrequent cause of external ocular infections. Patients typically have underlying ocular risk factors or are medically debilitated.¹ We describe a case of neonatal conjunctivitis due to MRSA and discuss its implications.

Case report

A 7 day old neonate was referred to the ophthalmology team with a day's history of purulent right conjunctivitis. The baby had been born at term by normal vaginal delivery; however the amniotic membrane had ruptured 48 hours prematurely. The mother had been started on amoxicillin and admitted to the maternity unit 24 hours after delivery. There was no outbreak of MRSA in the maternity unit at the time.

A conjunctival swab taken before antibiotic treatment grew MRSA. A repeat swab also grew MRSA which was sensitive to chloramphenicol. Further swabs grew MRSA from the nose and umbilicus. Both parents were doctors; one in hospital practice and the other in general practice. They were screened and found to be colonised with MRSA; the mother's low vaginal swab and nasal swabs were positive as was the father's nasal swab. The baby responded to chloramphenicol eye drops and the conjunctivitis resolved within a week. In addition, all three family members were treated with nasal mucopirocin and chlorhexidine skin wash. Post-treatment screening swabs showed the whole family was clear of MRSA.

Comment

The prevalence of MRSA carriage in health-care workers has been reported at between 0% to 36%, depending on the specialty.² The same study demonstrated a 40% transmission rate of MRSA from carrier healthcare workers to their households. Kniel *et al* found that eradication of MRSA was more difficult to achieve where household contamination had occurred, possibly because of recolonisation of the healthcare worker. Eradication was defined as clear swabs at 3 months post-eradication therapy.³

Peacock *et al*⁴ prospectively looked at determinants of acquisition and carriage of *Staphylococcus aureus* in infancy. They found a 68% concordance for *S aureus* carriage between mothers and infants. The fact that the bacterial strains were often the same in mother-infant pairs led them to conclude that the mother is the usual source; the strain acquired is dictated by environmental factors. Similarly, the case described is likely to be the result of maternal transmission of MRSA manifesting as conjunctivitis—both parents